publication on August 16, 1995 (60 FR 42482), is corrected as follows:

§ 230.17 [Corrected]

Paragraph 1. On page 42487, in the third column, in § 230.17, paragraph (a), line 5, is corrected by adding after the word ''A'', ''report is required when the individual's total earnings or wages'', before the word ''exceed''.

Dated: August 16, 1995.

By authority of the Board.

For the Board,

Beatrice Ezerski,

Secretary to the Board.

[FR Doc. 95–21073 Filed 8–23–95; 8:45 am]

BILLING CODE 7905–01–M

DEPARTMENT OF AGRICULTURE

Forest Service

36 CFR Part 242

DEPARTMENT OF THE INTERIOR

Fish and Wildlife Service

50 CFR Part 100

Alaska Federal Subsistence Regional Advisory Council Meetings

AGENCIES: Forest Service, USDA; Fish and Wildlife Service, Interior.

ACTION: Notice of meetings.

SUMMARY: This notice informs the public of the Regional Council meetings identified above. The public is invited to attend and observe meeting proceedings. In addition, the public is invited to provide oral testimony before the Councils on proposals to change Subsistence Management Regulations for Public Lands in Alaska as set forth in a proposed rule on August 15, 1995 (60 FR 42085–42130).

The following agenda items will be discussed at each Regional Council meeting: Introduction of Regional Council members and guests; election of officers; old business; new business: agency reports; review and development of proposals to change Subsistence Management Regulations for Public Lands in Alaska; and annual report.

DATES: The Federal Subsistence Board announces the forthcoming public meetings of the Federal Subsistence Regional Advisory Councils (Regional Councils). The Regional Council meetings may last two-three days and will be held in the following Alaska locations, starting on the date indicated.

Region 1 (Southeast)—Klawock—September 28

Region 2 (Southcentral)—Anchor Point—September 27

Region 3 (Kodiak/Aleutians)—King Cove—October 5

Region 4 (Bristol Bay)—Dillingham—October 10

Region 5 (Yukon-Kuskokwim Delta)—Bethel—October 3

Region 6 (Western Interior)—Aniak—October 10

Region 7 (Seward Peninsula)—Nome—October 26

Region 8 (Northwest Arctic)—Kotzebue—October 12

Region 9 (Eastern Interior)—Fairbanks—October 4

Region 10 (North Slope)—Anchorage—October 16

Notice of specific times and locations will be placed in local and statewide newspapers and on local radio stations.

FOR FURTHER INFORMATION CONTACT: Chair, Federal Subsistence Board, c/o Richard S. Pospahala, Office of Subsistence Management, U.S. Fish and Wildlife Service, 1011 E. Tudor Road, Anchorage, Alaska 99503; telephone (907) 786–3467. For questions related to subsistence management issues on National Forest Service lands, inquires may also be directed to Ken Thompson, Regional Subsistence Program Manager, USDA, Forest Service, Alaska Region, P.O. Box 21628, Juneau, Alaska 99802–1628; telephone (907) 586–7921.

SUPPLEMENTARY INFORMATION: The Regional Councils have been established in accordance with Section 805 of the Alaska National Interest Lands Conservation Act, Public Law 96–487, and Subsistence Management Regulations for Public Lands in Alaska, subparts A, B, and C (57 FR 22940–22964). The Regional Councils advise the Federal Government on all matters related to the subsistence taking of fish and wildlife on public lands in Alaska and operate in accordance with provisions of the Federal Advisory Committee Act. The identified Regional Council meetings will be open to the public. The public is invited to attend these meetings, observe the proceedings, and provide comments to the Regional Councils.

Dated: August 18, 1995.

Mitch Demientieff,

Chair, Federal Subsistence Board.

[FR Doc. 95–21010 Filed 8–23–95; 8:45 am]

BILLING CODE 3410–11–P; 4310–55–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 372

[OPPTS–400094; FRL–4954–6]

Toxic Chemical Release Reporting; Community Right-To-Know; Denial of Petition

AGENCY: Environmental Protection Agency (EPA).

ACTION: Denial of Petition.

SUMMARY: EPA is denying a petition to delete manganese and manganese compounds contained in iron-making and carbon steel making slags from the list of toxic chemicals subject to section 313 of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA). This action is based on EPA's conclusion that manganese and manganese compounds in slags do not meet the EPCRA section 313(d)(3) deletion criteria.

FOR FURTHER INFORMATION CONTACT: Maria J. Doa, Petitions Coordinator, 202–260–9592, e-mail: doa.maria@epamail.epa.gov, for specific information on this Denial of Petition, or for more information on EPCRA section 313, the Emergency Planning and Community Right-to-Know Hotline, Environmental Protection Agency, Mail Code 5101, 401 M St., SW., Washington, DC 20460, Toll free: 1–800–535–0202, in Virginia and Alaska: 703–412–9877 or Toll free TDD: 1–800–553–7672.

SUPPLEMENTARY INFORMATION:

I. Introduction

A. Statutory Authority

This action is issued under sections 313(d) and (e)(1) of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA), 42 U.S.C. 11023. EPCRA is also referred to as Title III of the Superfund Amendments and Reauthorization Act of 1986 (SARA) (Pub. L. 99–499).

B. Background

Section 313 of EPCRA requires certain facilities manufacturing, processing, or otherwise using listed toxic chemicals to report their environmental releases of such chemicals annually. Beginning with the 1991 reporting year, such facilities also must report pollution prevention and recycling data for such chemicals, pursuant to section 6607 of the Pollution Prevention Act of 1990 (PPA), 42 U.S.C. 13106. Section 313 established an initial list of toxic chemicals that was comprised of more than 300 chemicals and 20 chemical categories. Section 313(d) authorizes

EPA to add or delete chemicals from the list, and sets forth criteria for these actions. EPA has added and deleted chemicals from the original statutory list. Under section 313(e), any person may petition EPA to add chemicals to or delete chemicals from the list. EPA must respond to petitions within 180 days either by initiating a rulemaking or by publishing an explanation of why the petition is denied.

EPA issued a statement of petition policy and guidance in the **Federal Register** of February 4, 1987 (52 FR 3479), to provide guidance regarding the recommended content and format for submitting petitions. On May 23, 1991 (56 FR 23703), EPA published guidance regarding the recommended content of petitions to delete individual members of the section 313 metal compound categories. EPA has also published a statement clarifying its interpretation of the section 313(d)(2) criteria for adding and deleting chemical substances from the section 313 list (59 FR 61439, November 30, 1994).

II. Description of Petition

The American Iron and Steel Institute (AISI) petitioned the Agency on October 20, 1993, to qualify the listings of manganese and manganese compounds to exempt reporting of these substances when they are contained in slag generated from iron and carbon steel manufacturing operations. AISI (the petitioner) claims that, due to the tightly bound nature of the manganese-slag complex, the complex is relatively inert and does not present an unreasonable risk to human health or the environment. Moreover, the petitioner asserted that the manganese ion is not available to be leached from the complex due, again, to its tightly bound nature.

III. EPA's Technical Review of the Petition

The technical review of the petition to delete manganese and manganese compounds contained in iron-making slags and carbon steel-making slags included an analysis of the toxicological effects of manganese compounds as contained in the aforementioned slags. Based on the guidance published by EPA on petitions to delist individual members of the metal compound categories (56 FR 23703, May 23, 1991), EPA also reviewed the toxicity of manganese ion, as well as the availability of the ion from the aforementioned slags, (Refs. 1, 2, 3, and 4).

A. Chemistry Profile

1. *Manganese ion*. Manganese is a naturally occurring substance found in many rocks and as a constituent in several freshwaters and the ocean. Although pure manganese is silvery, much like iron in its appearance, manganese is rarely found in its pure state. Generally, it exists combined with other chemicals (such as oxygen, sulfur, and chlorine) (Ref. 5). As present in the slag, manganese is typically found as oxides and are relatively insoluble compounds.

2. *Manganese in slags*. Although manganese can be added directly into the iron and steel manufacturing process, generally the manganese found in the slags originates from iron ore. Slags containing manganese compounds can be generated from three processes: blast furnace; basic oxygen furnace; and electric arc furnaces. Slags are produced as the lighter fraction in each of the processes and are separated during the tapping procedure. After separation, the slag is cooled with water sprays and broken into smaller pieces. These smaller pieces are generally loaded in a truck for transport to an on-site landfill.

The slag may be used in concrete manufacture, as roadbed fill, as railroad ballasts, and as fertilizer components.

B. Toxicological Evaluation of Manganese Ion

It is generally recognized that manganese uptake and elimination are under homeostatic control, allowing for a wide range of dietary intakes considered to be safe. Further, manganese is an essential element, being required for normal human growth and maintenance of health (Refs. 3 and 4).

It has been reported that the average daily dose of manganese in the United States, England, and Holland ranges from 2.3 to 8.8 milligrams per day (mg/day). The Food and Nutrition Board of the National Research Council has determined a safe level of intake of manganese to be 2 to 5 mg/day for adults. In the normal adult, approximately 3 to 10 percent of dietary manganese is absorbed. However, dietary deficiencies of calcium and iron can increase that percentage. Therefore, it appears as if certain subpopulations, such as children, individuals with dietary deficiencies, pregnant women, and the elderly, may have an increased potential for heightened body burdens of manganese (Refs. 3, 4, and 6).

Manganese has been shown to readily penetrate the bloodbrain and placental barriers (Refs. 3 and 4). These findings are significant with respect to the well-known effects of manganese on the central nervous system (CNS) of adult humans and, probably, developing humans. Manganese elimination from the body is slow, and the clearance half-time from the brain is considerably longer than that for the whole body (Ref. 6).

1. *Acute toxicity*. In 1984, the Agency generated a comprehensive health assessment for manganese in which median lethal dose (LD_{50}) values for several inorganic manganese compounds were calculated. These values range from 400 to 830 milligrams per kilogram (mg/kg) by the oral route and 38 to 64 mg/kg by parenteral injection (Ref. 6).

2. *Neurotoxicity*. The CNS effects of manganese compounds have long been known. The first medical description of chronic manganese neurotoxicity (manganism) in workers is generally credited to Couper in the 1830s (Ref. 6). The disorder, manganism, has been described in workers in industries that typically involve exposure to manganese oxide fumes. Such industries include: Ore crushing; ferroalloy production; steel making; dry cell battery manufacture; and, welding rod manufacture. Those who develop chronic manganese poisoning initially exhibit a hyperactive maniacal state that progresses through lassitude and weakness to a later stage characterized by parkinsonism, dystonia, and cerebellar ataxia. Although the course and degree of manganese intoxication can vary greatly among individuals, the chronic state can develop without an initial manic state. However, once the chronic stage has developed, the neurologic dysfunction is irreversible (Ref. 6).

There is evidence of neurotoxic effects in adult humans and animals. These effects are also a probable hazard to human fetal and neonatal nervous systems (i.e., developmental neurotoxicity) based on circumstantial human data and on test data in animals. There is also human and animal evidence of acute toxicity (manganese pneumonia, metal fume fever in humans, severe lung damage in animals) and human and animal data on chronic pulmonary effects (Ref. 6).

Several studies have noted neurotoxic effects from soluble forms of manganese. As specified in the Integrated Risk Information System (IRIS) and other sources, neurotoxicity is the critical endpoint of concern. There are two epidemiological studies describing toxicologic responses in humans from excess amounts of manganese dissolved in drinking water (Ref. 6). The first, Kondakis et al. (1989) studies three

areas in northwest Greece (Ref. 6). The total population of the three areas (A, B, C) studied ranged from 3,200 to 4,350 people and manganese concentration in well water ranged from 3.6 micrograms per litre (ug/1) to 2300 ug/1. Individuals chosen for the study were submitted to neurological examination; whole blood and hair manganese concentration were also determined. The concentration of manganese in the whole blood did not differ between the three areas, but this is not considered to be a reliable indicator of manganese exposure. However, there was a significant difference noted in neurological scores for area C versus area A even when both age and sex are taken into account. A lowest observed adverse effect level (LOAEL) of 0.06 mg Mn/kg-day and a no observed adverse effect level (NOAEL) of 0.005 mg Mn/kg-day for the study were estimated from concentrations using default values (a water consumption of 2 litres/day, and a 70 Kg assumed adult body weight) (Ref. 6).

The second report is by Kawamura et al. (1941) and is the only epidemiological study describing toxicologic responses in humans consuming large amounts of manganese in drinking water (Ref. 6). Twenty-five cases of manganese poisoning were reported, with symptoms including lethargy, increased muscle tonus, tremors and mental disturbances. Elderly people showed the most severe symptoms. Although the intake of manganese in the diet was not determined, the approximate intake estimated for the study was 0.8 mg/kg-day. This supports the LOAEL estimated from the Kondakis et al. (1989) study (Ref. 6). It should be noted that the well water in the study was contaminated with zinc, and that this could have effected the results. The impacts of the zinc contamination were not evaluated.

Use of the Greek study is supported upon review in context of additional information. The spectrum of neurological dysfunction observed in chronic manganese neurotoxicity effects in humans can be reproduced, in part, in different animal species, including rats, rabbits, and monkeys (characteristic CNS signs were produced in monkeys exposed to manganese dioxide) (Ref. 6).

Roels et al. (1992) reported that workers who had chronically been exposed to manganese (0.215 mg manganese/m^3) for respirable dust and 0.948 mg manganese/m^3 for total dust with a duration of employment ranging from 0.2 to 17.7 years) performed worse than controls on several measures of neurobehavioral function (such as visual reaction time, eye-hand coordination, uncertainty, etc.) (Ref. 6). A LOAEL of 0.05 mg/m^3 was derived from the study. A previous study performed by Roels et al. (1987) found significant differences in mean scores between manganese-exposed and referenced subjects for visual reaction time, eye-hand coordination, hand steadiness, and audio-verbal short-term memory (Ref. 6). Total airborne manganese dust ranged from 0.07 to 8.61 mg/m^3 for a duration of employment spanning from 1 to 19 years. During the study it was also noted that there were a significantly greater prevalence of coughs during the cold season and episodes of acute bronchitis in the manganese-exposed group. A LOAEL of 0.34 mg/m^3 was derived from the study (Ref. 6).

As noted in IRIS (November 1993), there is a consistent pattern of evidence indicating that neurotoxicity is associated with low-level occupational manganese exposure (Ref. 6). More detail on the neurotoxic effects observed from chronic exposure to manganese is given above.

3. *Respiratory toxicity*. As specified in IRIS (November 1993), as a route of exposure, the respiratory tract is the most important route of entry (Ref. 6). Particles which deposit in the extrathoracic and tracheobronchial regions (greater than 2.5 micrometers (um)) are predominantly cleared by the mucociliary escalator into the gastrointestinal tract where absorption is low. Smaller mode particles (greater than 2.5 um) are deposited in the pulmonary region where 100 percent absorption is assumed. However, some researchers have suggested that neurotoxic metals can be directly transported to the brain olfactory bulbs (Ref. 6).

After absorption by the respiratory tract, manganese is transported directly to the brain via the blood stream, bypassing the liver. This direct path has been suggested to account for the difference in toxicity between inhaled and ingested manganese (Ref. 6).

4. *Reproductive/developmental toxicity*. There is insufficient information on the developmental toxicity of manganese by inhalation exposure, and the same is true for information on the female reproductive function. The study of the female reproductive toxicity of inhaled manganese in males also needs to be characterized more fully (Ref. 6).

5. *Carcinogenicity*. Manganese has been identified as Class D or not classifiable as to human carcinogenicity. Existing studies are inadequate to assess the carcinogenicity of manganese (Ref. 6).

6. *Ecological effects*. Manganese ion exhibits a moderate toxicity to aquatic and terrestrial organisms and has a high potential to bioaccumulate. Manganese is an essential tract element or micronutrient for microorganisms, plants and animals. It is a functional component of nitrate assimilation, in the Hill reaction of photosynthesis, and is an essential catalyst of many enzyme systems.

Acquatic chronic toxicity values are as low as 3.2 to 5.7 parts per million (ppm) for invertebrates and as low as 12 ppm for fish. Concentrations as low as 0.2 to 0.3 ppm were toxic to some marine algae. Aquatic chronic toxicity data are more limited. The no observed effect concentration (NOEC) for rainbow trout eggs exposed to manganese for 29 days is less than 370 parts per billion (ppb). The lowest observed effect concentration (LOEC) in this study was calculated to be approximately 370 ppb (Ref. 7).

Marine plants and animals may bioaccumulate manganese; bioconcentration values have been reported to be approximately 3,000. Furthermore, bioconcentraton values for shellfish range from 1,000 to 10,000; and for fish, marine algae, and plants, from 100 to 100,000 (Ref. 7).

C. Toxicological Evaluation of Manganese in Slags

1. *Human health effects*. The Agency has identified some potential hazards resulting from exposure to the manganese-slag complex. Generally, these hazards are associated with the slag in a granular or powdered form and are consistent with typical concerns of particulate exposure. These include: Eye irritation; lung overload; and lung irritation. The insolubility of the manganese-slag complex allays most systemic toxicity concerns with the exception of lung overload. The Agency does not consider the hazard of lung overload to be significant (Refs. 3 and 4).

2. *Ecological effects*. Manganese levels in leachate from slags as reported in the petition exceed the range of manganese reported in most natural freshwaters. The upper leachate level reported in the petition ranged from 28 to 32 ppm, with averages as high as 7 and 11 ppm. Manganese concentrations in natural freshwaters around the world normally range from 10 to 850 ppb, with an average of 35 ppb. However, some reservoirs may have concentrations of up to 150 ppm; subsurface and acid mine waters may contain 10 ppm (Ref. 7).

The petitioner contends that ''manganese compounds in slags do not

dissociate or react to yield metal ions because the metal ion is tightly bound in a calcium-silica matrix and cannot be released.'' However, this conclusion is inconsistent with the information from other studies presented in the petition indicating high levels of manganese from leaching are possible.

D. Availability of Manganese ion from Slags

Although it is established that leaching of manganese from the slag occurs, there is insufficient information regarding the ultimate fate of the leachate for a detailed characterization. A variety of conditions (i.e., geology, pH, soil organic content, etc.) combine in a complex manner to severely limit modeling of the fate of the leachate.

Manganese may be leached from slags under acidic and reducing conditions, which are the conditions expected to prevail in landfilled slags that are in contact with the aquatic environment. Further, these same conditions are conducive to reduction of the manganese oxides normally found in slags to the water soluble manganous ion, (Mn^{+2}). Although Mn^{+2} often precipitates with carbonate ions as $MnCO_3$, this is not always the case, and various lines of evidence suggest that Mn^{+2} may enter ground water supplies and/or may reach surface waters. Evidence also shows that sorption of manganese to soil is highly variable, and that release may actually occur under certain conditions (Ref. 1). Thus, it cannot be concluded that ''any manganese leached from slags is quickly adsorbed by the surrounding soil'' as the petitioner claims.

The petitioner reports the slag to have a pH of 9 to 11 in which the manganese is present in an insoluble oxide form. Slag piles are generally fully exposed to weather conditions and are present in a wide range of sizes, very small particulates to large blocks. Under acidic conditions, such as those present in acid rain (pH 5.5), the predominant species of manganese is not the insoluble oxide form but the soluble ion form, manganese^{+2}. The petitioner also reports a range of manganese leachate measured from a variety of slag sources; the upper level being 22 to 32 mg/1 (ppm) of manganese ion (Refs. 1 and 6).

The soluble manganese ion can then hydrolyze, form insoluble oxides, exist as Mn^{+2} in solution, precipitate with carbonates and other anions, and form insoluble sulfides depending on the redox potential of the water media, pH, temperature, and the mix of anions present. Most of these reactions are catalyzed by biota. Adsorption of Mn^{+2} is favored in soils with a large percentage of clay particles and organic material. Anaerobic conditions and acidified conditions favor resolubilization of Mn^{+2} (Refs. 1 and 6).

E. Technical Summary

EPA's toxicological evaluation of manganese ion indicates that manganese can cause neurotoxic effects in humans, exhibits moderate toxicity to aquatic and terrestrial organisms, and has a high potential to bioaccumulate. EPA's assessment of the availability of manganese ion from iron-making and carbon steel-making slags indicates that a wide range of manganese leachate from slag piles has been documented (noted in the petition). This indicates that leaching of the manganese ion is expected. Measured leachate levels, as specified in the petition, exceed acute and chronic aquatic toxicity values and those reported as toxic to certain plants. Evidence also shows that sorption of manganese to soils is highly variable, and that release may actually occur under certain conditions (Refs. 1, 6, and 7).

IV. Rationale for Denial

EPA is denying the petition to delete manganese and manganese compounds in iron-making and carbon steel-making slag from the EPCRA section 313 list. EPA believes that manganese ion can become available at levels which can reasonably be anticipated to induce adverse human health and environmental effects. EPA believes that manganese and manganese compounds in iron-making and carbon steel-making slag meet the toxicity criteria of EPCRA section 313(d)(2)(B) based on available neurotoxicity data, and that they meet the toxicity criteria of EPCRA section 313(d)(2)(C) based on the available acute environmental toxicity and bioconcentration data.

V. References

(1) USEPA/OPPT, Boethling, Bob, *Environmental Fate of Manganese* dated January 18, 1994.

(2) USEPA/OPPT, Macek, Greg, *Final Report: Engineering Support for EPA Review of Section 313(e) Petition on Manganese and Manganese Compounds in Iron-Making and Carbon Steel-Making Slags* dated January 27, 1994.

(3) USEPA/OPPT, Murphy, James J., *Preliminary Review of Systemic Toxicity for EPCRA Section 313 Delisting Petition on Manganese and its Compounds in Slags* dated November 19, 1993.

(4) USEPA/OPPT, Murphy, James J., *Review of Systemic Toxicity of Manganese with Particular Reference to Manganese-Containing Slag* dated December 29, 1993.

(5) USEPA/OPPT, Rakshpal, Ram, *Section 313(e) Petition on Manganese and Manganese Compounds in Iron-Making Slags and Carbon Steel-Making Slags (Chemistry Report)* dated December 9, 1993.

(6) USEPA/OPPT, Rusak, Linda, *Technical Integrator Report* dated April 1995.

(7) USEPA/OPPT, Smerchek, Jerry C., *Ecological Hazard Review of the American Iron and Steel Institute Petition to Delist Manganese and Manganese Compounds Contained in Iron-Making Slags and Carbon Steel-Making Slags* dated December 9, 1993.

VI. Administrative Record

The record supporting this denial of petition is contained in the docket number OPPTS–400094. All documents, including an index of the docket, are available in the TSCA Nonconfidential Information Center (NCIC), also known as the TSCA Public Docket Office, from noon to 4 p.m., Monday through Friday, excluding legal holidays. The TSCA Public Docket Office is located at EPA Headquarters, Rm. NE–B607, 401 M St., SW., Washington, DC 20460.

List of Subjects in 40 CFR Part 372

Environmental protection, Chemicals, Community right-to-know, Reporting and reccordkeeping requirements, and Toxic chemicals.

Dated: August 15, 1995.

Lynn R. Goldman,

Assistant Administrator for Prevention, Pesticides and Toxic Substances.

[FR Doc. 95–21039 Filed 8–23–95; 8:45 am]

BILLING CODE 6560–50–F

FEDERAL COMMUNICATIONS COMMISSION

47 CFR Part 73

[MM Docket No. 95–134, RM–8679]

Radio Broadcasting Services; Sanford, NC

AGENCY: Federal Communications Commission.

ACTION: Proposed rule.

SUMMARY: The Commission requests comments on a petition filed by Woolstone Corporation seeking the allotment of Channel 276A to Sanford, NC, as the community's second local FM service. Channel 276A can be allotted to Sanford in compliance with